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AN 2006:44325 CAPLUS
DN 145:139583
TI Specific protein interaction of human Pag with Omi/HtrA2 and the
activation of the protease activity of Omi/HtrA2
AU Hong, Seung-Keun; Cha, Mee-Kyung; Kim, Il-Han
CS Department of Biochemistry, Paichai University, Taejon, 302-735,
S. Korea

SO Free Radical Biology & Medicine (2006), 40(2), 275-284

CODEN: FRBMEH; ISSN: 0891-5849

PB Elsevier

DT Journal

LA English

AB The human PAG gene product (hPag), one member of the TSA/Ahpc family, is

overexpressed by oxidative stress, which causes apoptosis. To investigate

the apoptotic signal transduction mediated by hPag, hPag-binding protein

was screened using the yeast two-hybrid system.

Omi/HtrA2 was identified as the hPag-binding protein.

Omi/HtrA2, a potent

proapoptotic factor, is released from the mitochondria into the cytoplasm

as the mature form showing serine protease activity

during apoptosis in response to oxidative stress. We found that

hPag was

able to interact with the mature form of Omi/HtrA2, not with the precursor

form of Omi/HtrA2. The binding of Omi/HtrA2 to hPag was shown to involve

the PDZ-binding domain in Omi/HtrA2. Also, the carboxyl-terminal domain

of hPag was shown to be critical for the protein interaction.

Using the

yeast two-hybrid system and in vitro binding assay,

the reduced form of hPag was able to interact with Omi/HtrA2.

Interestingly, the protease activity given by the

mature form of Omi/HtrA2 was significantly activated by the

binding to

hPag. Taken together, these results suggest that the specific protein

interaction may participate as a mol. switch in modulating cell death in

response to oxidative stress.

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DN PubMed ID: 15101818

TI Binding of proteins to the PDZ domain regulates proteolytic activity of HtrA1 serine protease.

AU Murwantoko; Yano Masato; Ueta Yoshifumi; Murasaki Ai; Kanda Hidenobu; Oka

Chio; Kawaichi Masashi

CS Division of Gene Function in Animals, Nara Institute of Science and

Technology, 891605 Takayama, Ikoma, Nara 630-0101, Japan.

SO The Biochemical journal, (2004 Aug 1) Vol. 381, No. Pt 3, pp. 895-904.

Journal code: 2984726R. E-ISSN: 1470-8728.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
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LA English

FS Priority Journals

EM 200412

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AB HtrA1, a member of the mammalian HtrA (high temperature requirement A)

serine protease family, has a highly conserved protease domain followed by a PDZ domain. Accumulating evidence has indicated that PDZ domains regulate protease activity of HtrA proteins. We searched for binding partners of the PDZ domain of

mouse HtrA1 by yeast two-hybrid screening, and isolated proteins that were recognized by the HtrA1 PDZ domain through

their C-terminal ends with a core consensus

Phi-X-Phi-[V/L/F/A]-COOH

sequence (where Phi is a hydrophobic/non-polar amino acid).

C-propeptides

of fibrillar collagens were most frequently isolated. Type III procollagen alpha1 C-propeptide, which was used as a model protein, was

digested by HtrA1. HtrA1 cleavage of the collagen C-propeptide was

enhanced by reductive denaturation of the C-propeptide and partly inhibited by removal of the C-terminal four amino acids from the C-propeptide, suggesting that the substrate recognition was facilitated by

the binding of the free C-terminal ends of substrates to the PDZ domain of

HtrA1. The synthetic oligopeptide (GM130Pep) that fitted the consensus

recognition sequence bound to HtrA1 with a high affinity

(K(d)=6.0 nM).

GM130Pep stimulated HtrA1 protease activity 3- to

4-fold, but did not efficiently stimulate the activity of an

HtrA1 mutant

lacking the PDZ domain, supporting the notion that the PDZ domain enhances protease activity upon ligand binding

. The peptide derived from Type III collagen alpha1 C-propeptide specifically stimulated protease activity of HtrA1,

but did not stimulate nor significantly bind to HtrA2,

suggesting that the

collagen C-propeptide is a specific physiological regulator of HtrA1.



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